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THE
LARYNGOSCOPE.

VOL. LX JUNE, 1950. No. 6

STRUCTURAL CHANGES IN THE CILIATED EPITHELIAL CELL DURING UPPER RESPIRATORY INFECTION: PRELIMINARY REPORT.

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and
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During the routine study of nasal smears we thought it would be interesting to stain the secretions of the common cold according to Dr. George N. Papanicolaou's method.[1,2] His stain has proved particularly suitable for epithelial cells in differentiating malignant changes. It seemed reasonable that such a stain would give additional information on changes which take place in the nasal epithelium during acute upper respiratory infections.

Nasal smears have been repeatedly studied with the more traditional stains, as Wright's, Giemsa's and Hansel's. In 1930 and 1934, Hilding[3] reported studies of the common cold in which he had observed large numbers of living epithelial cells and fragments of cells in the secretions during the first three days of a cold. In order to study the pathological changes he took biopsies from the noses of 30 volunteers suffering from colds in various stages. The most striking change was the extensive loss of epithelial cells, both of the columnar and of the deeper lying stellate types. After a few days the

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epithelium regenerated and resembled the normal controls. All specimens were stained for bacteria and carefully examined, but no bacteria were ever found to invade the epithelial cell.

The material for this study consists of numerous nasal smears taken at various times in the course of colds and of others taken regularly twice a day throughout the duration of the infection. To date four complete series and numerous incomplete series and isolated smears have been studied with Papanicolaou's stain, in all 75. Most of the smears were made by blowing the material from the nose, rather than by swabbing, and then spreading the material gently with a metal probe. Duplicate smears were made in each case in order to have a comparison with Wright's stain.

Ciliated epithelial cells appear in moderate numbers in the first smears taken, that is, within six hours of the onset of nasal secretion. Succeeding smears show an increasing number of these cells which reach a peak, depending upon the severity of symptoms, between the second and fifth days. The number then diminishes, the cells becoming very rare as the cold subsides.

The ciliated epithelial cells show definite degenerative changes in contrast to the normal ciliated epithelial cell.

Normal columnar ciliated cells appear in Fig. 1, obtained by swabbing from a normal nose. In the Papanicolaou stained smears the cilia are clearly and probably almost perfectly preserved—which is not true in smears stained with Wright's stain. The bluish-purple nucleus is finely granular and contains one to three nucleoli. There is a definite nuclear membrane. The nucleus is usually eccentrically oval and is placed at the narrow end of the cell away from the cilia. Normal ciliated bronchial epithelium with this stain is described in "The Cytological Diagnosis of Cancer" by the Vincent Memorial Hospital staff.[4] It appears very similar to the cells we have seen in nasal smears. The cytoplasm is lightly granular and palely acidophilic or basophilic in the normal cell. In well preserved cells the cilia are clearly visible at the broad end

of the cell; often the pink staining ciliary bodies can be seen. The cellular borders are distinct in the single exfoliated cells, but when they are in clumps they tend to be obscured. At times the cytoplasm is very elongated.

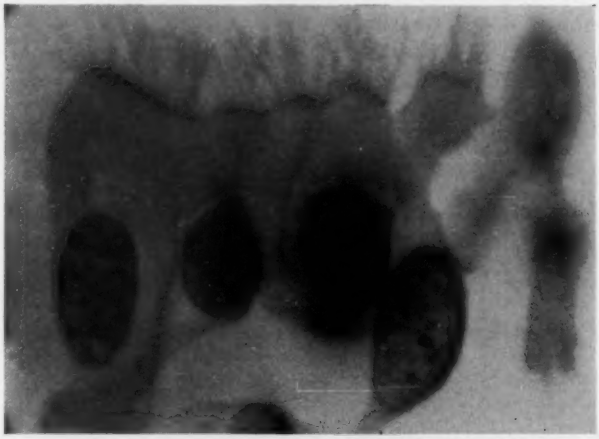

Fig. 1. A group of normal ciliated epithelial cells from a nasal smear.

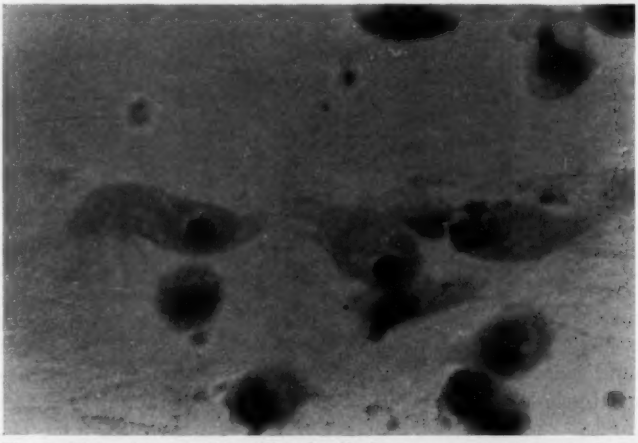

Fig. 2. Three degenerating ciliated cells from an acute upper respiratory infection. The left and middle cells have pyknotic nuclei, nuclear halo with two black bodies, and a pink cytoplasmic body. The cell on the right shows karyorrhexis. Magnification is about 950×.

Frequently normal goblet cells with ballooned vacuolated cytoplasm are identified. Squamous and basal epithelial cells are also normally identified and appear in varying numbers.

Fig. 3. Karyorrhexis with pink nuclear inclusion bodies (density in black and white is lighter than the masses of chromatin).

In contrast to the normal ciliated columnar exfoliated cell, the remaining illustrations show examples of degenerative changes which occur in these cells during a cold. The nuclear degeneration seems to fall into three groups according to the following patterns:

1. In the first there is a marginal arrangement of the basophilic chromatin on the nuclear membrane. The cytoplasm and cilia appear normal.

2. In the second the nucleus shows karyorrhexis with irregularly distributed pyknotic clumps of chromatin. These cells usually show a halo around the clumps. In between the dark masses of chromatin one or more pink bodies are occasionally found. The cytoplasm is more roughly granular and often contains one or more smooth, pink spherical bodies usually near the nucleus. The cilia appear normal. Some of the cells are elongated and constricted in the middle. This is more marked and numerous in the following pattern.

3. In the third the nucleus becomes completely pyknotic. Often it is seen as one solid, round, black mass. The eccentric position may or may not be maintained. About the chromatin mass there is frequently a rather clear halo which may contain one to three small black bodies more like granules of the same dark nuclear color. One is impressed with the frequency of this pattern. The smooth, pink spherical body or bodies in the cytoplasm mentioned in the second pattern are regularly present in this group. This group is most numerous in all cases thus far studied at the peak of the desquamation.

At the same time there are numerous "empty nuclei" in ciliated cells. These seem to have lost the black mass of chromatin material. There appears to be a clear area without visible structure. All manner of nuclear material including whole pyknotic nuclei, that is, the spherical black masses,

Fig. 4. Degenerating spherical cell. The ciliary bodies and cilia are quite prominent. Nucleus is pyknotic.

with or without halo and small black bodies, are seen scattered unattached over the slide.

The cytoplasm seems to fall into two patterns of degeneration. One is the shortening of the cell, some becoming almost spherical (see Fig. 4). The other pattern is of constriction

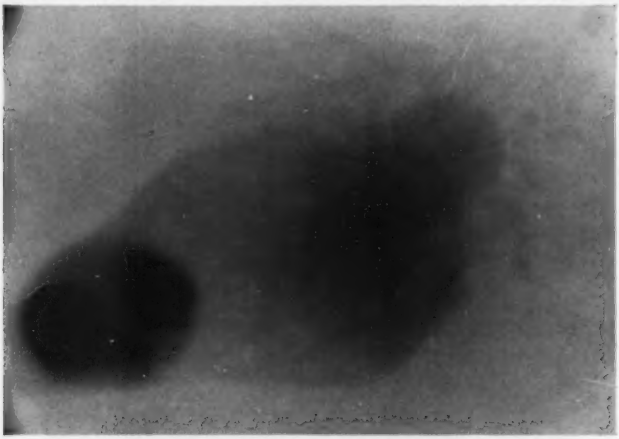

Fig. 5. Tufted ciliated cell. Nucleus shows karyorrhexis and pink body.

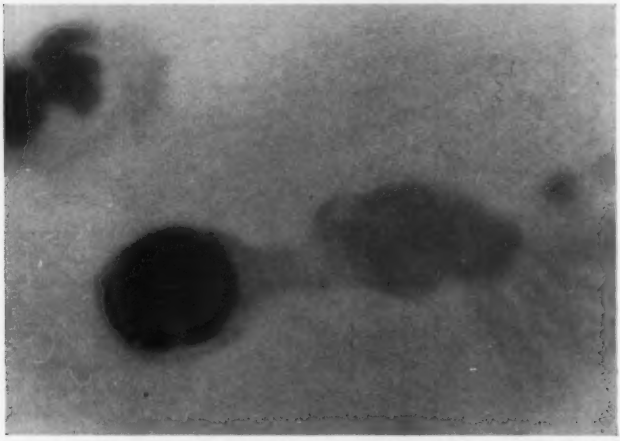

Fig. 6. Nuclear portion is beginning to separate and shows margination of chromatin.

and elongation, even to the point of breaking in half (see Fig. 6). The nucleus tends to be in one half, the bulk of the cytoplasm and cilia in the other. This seems to be a progressive process, as after these halves separate, many cellular

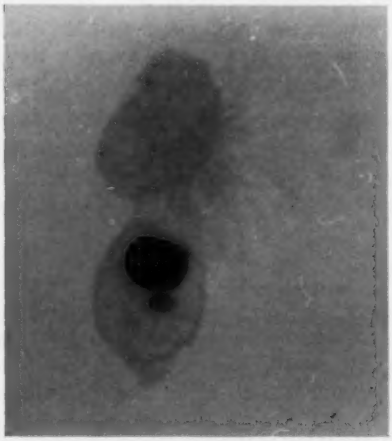

Fig. 7. Separation of nucleus and cytoplasmic portions is complete.

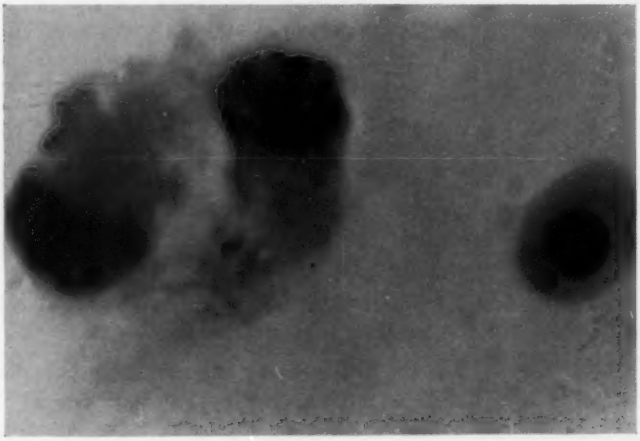

Fig. 8. Isolated nucleus to right. Two degenerating cells left showing karyorrhexis and center one margination.

fragments are present in the slide. Many tufts of cilia attached to small portions of cytoplasm without nucleus are seen. In both patterns the cilia may arise from the sides of the cell. This wide attachment of cilia has been observed in tissue cultures of ciliated epithelium.[5] In the more spherical they are seen attached to a large part of the surface. In these the nuclei appear more normal. The pink cytoplasmic body is found in the majority of all the degenerating ciliated cells.

In all these slides of acute colds very few bacteria are noted and they have never been seen within the ciliated epithelial cell or appearing to invade it. Evidently these epithelial cells degenerate even though the presence of bacteria is not evident.

DISCUSSION.

The Papanicolaou stain gives particularly clear nuclear detail which makes possible the determination of and the distinction between the various types and conditions of exfoliated epithelium; whereas Wright's, Giemsa's and Hansel's stains are especially good for showing eosinophiles. The epithelial cell is readily identified as such in these stains, but the nuclear detail and cilia are not clear;[3] nevertheless, in the smears stained lightly with Wright's one can identify with difficulty the same halo, pink and black nuclear and perinuclear bodies.

The consecutive series of smears are especially valuable in determining the timing of desquamation. In random or routine slides the epithelial cells were not always found in large numbers. The ciliated epithelial cells are rare in smears taken from subacute and chronic infections; but these structural modifications have been seen in all cases of acute upper respiratory infection thus far studied.

The stain is selective for the degenerating epithelial cells as they are easily spotted by their acidophilic reaction.

The significance of the intranuclear and cytoplasmic bodies, both pink and black, is unknown. Nevertheless, Dr. E. V. Cowdry and Dr. Walter Siebert, pathologist, have examined these slides and suggest a possible correlation of these changes

with the virus etiology of a cold. The marked progressive degeneration of both nucleus and cytoplasm is evidence of injury to the cell.[6]

SUMMARY.

By means of the Papanicolaou stain, degenerative structural changes have been shown to be present in the exfoliated ciliated epithelial cells in nasal secretions. Prominent among these are intracellular bodies of two kinds which to our knowledge have not previously been described. Their identification and significance at this time awaits further investigation.

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THE FINDING OF POSITIONAL NYSTAGMUS IN THE DIZZY PATIENT.*

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The complaint of severe, recurrent dizzy attacks that occur with a change of position of the head or body is very common in our practice. Positive clinical findings unfortunately are rarely found and the dizziness is attributed to a functional disorder or even malingering in those claiming dizziness following a head injury. Positional nystagmus, when found, is positive evidence of organic pathology that is causing the dizziness. It is easily seen, easy to test for and is an essential part of every complete otoneurological examination.

The primary purpose in presenting this paper is to describe positional nystagmus, and how to detect it in the dizzy patient. A relatively brief discussion will be given on the mechanism of positional nystagmus and the location of the pathology. Some of the interesting points and problems presented by these patients will be discussed. A movie of two of the patients will be shown.

Although we had a patient in 1935 who had severe positional nystagmus, no other cases came to my attention until two years ago. Since then I have seen 12 cases that showed very active positional nystagmus and presented problems that made it necessary for me to investigate the subject farther.

Cases that had vertigo and nystagmus when the position of their head was changed have been reported for many years. Bárány was one of the first to report cases as early as 1911.

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Although there has been some general discussion of its occurrence under the heading of physiology, pathology or mechanism of nystagmus, until recent years there has been very little written specifically on the subject of positional nystagmus. The textbooks usually mention only the fact that a change of position or posture may cause vertigo or nystagmus. Positional nystagmus is not listed in the index in many textbooks.

Most of the neurosurgeons and otologists who were asked about positional nystagmus were not familiar with it. Last year at this Section Meeting in Los Angeles I asked several of the members about it, and as very few of them had a speaking acquaintance with the term positional nystagmus, it seemed to me that this was a very appropriate subject to present at this meeting.

HOW TO TEST FOR POSITIONAL NYSTAGMUS.

Positional nystagmus is the nystagmus that is produced when the position of the patient's head is changed or when the head is in a certain posture. As no other stimulation is required, the tests are, therefore, very easily and simply done. If the patient gives the history of this type of dizziness, it might be well to test for positional nystagmus before proceeding with the usual otoneurological examination, since this finding is not constant, as will be explained later. The patient is put on a couch or table. At first he sits erect and then lies straight back to the supine position. This may cause the nystagmus. In others it will occur if they lie back with their heads to the right or to the left. Although some writers seem to stress the possible effect of torsion of the neck as the cause of this, it is my personal opinion that it is not important. If this were true we would have it very frequently when we consider that 150,000,000 people twist their necks all day and night. Some patients while in the supine position will rotate their head to the right or to the left and have positional nystagmus. Some patients become dizzy and have nystagmus when they sit up from the supine position or put their head back in the optimum position from the head erect position.

Other positions that can be tried are the head hanging position or stooping over position. In other words, any change of position of the head may elicit nystagmus and some patients develop the nystagmus in several of these position changes.

Positional nystagmus is an abnormal pathological finding and is accompanied by severe vertigo, sweating, pallor, etc. It occurs spontaneously in patients who have no other illness and in patients following a head injury as well as in patients with CNS pathology, such as brain tumors, multiple sclerosis, etc. It frequently is the only pathology found (by neurologists or otologists) to account for dizziness. In all of my cases there were essentially normal findings in the ear, nose and throat, including the audiometer test, the Romberg test and the caloric and rotation tests. There was no spontaneous nystagmus or pastpointing in the head erect position.

The nystagmus that occurs in the cases I have seen is very active and wild and of greater amplitude than that produced by caloric or rotation tests. Frequently the patient will shout, "There it goes!" and will grab hold of anyone or anything available, and they appear very frightened. They may close their eyes, but the eyes can be seen to twitch violently beneath the lids. Sometimes the nystagmus does not start for several seconds after the change of position. It may last a few seconds or it may continue for minutes. Many patients have complained of dizziness, but unless they had active nystagmus for several seconds I have not called them true positive cases of positional nystagmus.

What kind of nystagmus do you see when they are active? It seems to me that my cases have shown every type there is—horizontal, rotatory and vertical. They are so active and wild that many times I have not been able to tell exactly in what direction the quick component moves. When I first started having movies taken of these cases, about a year ago, I had hoped to have high speed movies made in order to help me analyze just what their eyes were doing. I took six of my patients to the USN Hospital at Oakland to have movies taken in their photographic laboratory. The movies have been one disappointment after another. Either the patient could

not be made to produce nystagmus after repeatedly changing his position or else the camera did not perform when the nystagmus was active — the camera was not wound up, it broke down or was out of focus, etc., at the crucial moment. Finally with the aid of Dr. John Howard, who has a new Bolex camera, and the help of a patient who performed regularly, we were able to get one good film. This film shows a definite vertical nystagmus, whereas my notes showed that I had considered it "marked rotatory nystagmus, with a vertical element." Having seen this movie, I am not sure that some of the other cases that I called rotatory would not have shown more of a vertical character in a movie.

Although the nystagmus may occur in any direction, or even may change direction during an attack, it is very important to detect the quality, character and direction of the nystagmus in order to interpret the mechanism of this phenomenon. This is particularly true if there is a vertical nystagmus, for we have always considered spontaneous vertical nystagmus to be of central origin.

When do you make the tests for positional nystagmus? Whenever the patient describes his dizziness as coming on when he changes his position, you should make tests for positional nystagmus. It must be very strongly emphasized here that you may have to repeat the test several times to elicit positional nystagmus. This was forcefully impressed upon me by my first patient who was sent to me by an insurance company before appearing before the Industrial Accident Commission. He had a fractured skull with unconsciousness 20 months before. Since his injury he complained of occasional severe dizziness when he moved his head. Two well known neurosurgeons had never found organic pathology to account for his dizziness. My otoneurological examination was completely negative. Once, when he put his head in the optimum position, he said he was dizzy, but there was no nystagmus. I had him lie down and change his position in various ways to make him dizzy. The following day I repeated the examination, which was again normal. I then explained to the patient that I could find nothing organic to account for

his dizziness and although I did not doubt that he felt dizzy, I would have to send a report of normal findings to the insurance company. The patient remonstrated so loudly and sincerely, while I was trying to usher him out of the office, that I consented to test him once more. Again I had him recline from the erect sitting position to the supine position and he suddenly yelled, "There she goes!" He was right. He had a wild rotatory nystagmus to the right that lasted 15 seconds. Repeating this procedure twice produced no dizziness or nystagmus but the next attempt produced dizziness and nystagmus. The necessity for repeated testing has also been impressed on me in my attempt to get movies of these patients, as previously discussed. You do *not* get positional nystagmus every time you test the patient.

It should be emphasized that if positional nystagmus is found, it is positive evidence of organic pathology that is causing the vertigo and it eliminates the diagnosis of functional basis for the dizziness. This is very important for all patients, and particularly important for the post-traumatic industrial case. If you do not find nystagmus it does not necessarily mean that the patient has not had, nor will not have, positional nystagmus; also, it should be emphatically stated that any otologist who does not make the test for positional nystagmus on two or three occasions on a patient who says he is dizzy with a change of position is not making a complete otoneurological examination. I am emphasizing the necessity of careful testing, repeated if necessary, for I am sure the chief reason that we all have failed to see more of these cases is the fact that we have not tested sufficiently for it.

THE MECHANISM OF POSITIONAL NYSTAGMUS.

Bárány proposed the theory that the otolith system was concerned with static equilibrium, vertigo and nystagmus, and felt that positional nystagmus was due to disturbance in the otoliths. A great deal of research has been done and many articles written upon the entire subject of the complicated mechanism of nystagmus. The utricle was attributed

by many to be the site of origin of positional nystagmus. As you all know, there is a considerable difference of opinion on many aspects of nystagmus. It seems to me that it is not advisable to enter into this controversy at this time. Instead of this I am going to give two references that I feel are outstanding, and I am going to quote briefly from each, for I believe that they bring this subject up to date in the best possible manner.

The first reference is to McNally's article entitled, "The Physiology of the Vestibular Mechanism in Relation to Vertigo."[1] After reviewing the literature and experimental work, McNally said: "Such well controlled experiments as these do not indicate that nystagmus arises from utricular stimulation." McNally also said: "Nylen and Lindsay have classified positional nystagmus into type I and type II. In type I, the direction of the nystagmus changes with a change of position of the head. It occurs predominantly in CNS lesions. In type II, the direction of the nystagmus does *not* change with changes in position of the head; but the nystagmus may appear only in certain positions or it may be influenced in intensity by the position of the head. It may be found *either* in peripheral lesions or in central lesions." "It has not been definitely determined whether the cause of positional nystagmus is a lesion within the labyrinthine end organs or whether it is the result of some lesion within the CNS."

The other important reference is to the papers by Dr. J. R. Lindsay.[2,3] He writes: "Observations on known central lesions such as tumors, multiple sclerosis and cerebral concussion agree that a positional nystagmus type I invariably indicates a central location, whereas type II may occur either in a peripheral or central lesion." "Positional nystagmus is a relatively infrequent occurrence in lesions known to be situated in the peripheral ear." "It was demonstrated infrequently in the Ménière's disease group."

My own opinion is that positional nystagmus is usually due to central pathway pathology. The vertical nystagmus frequently seen in these cases and shown in the movies seems to be strong evidence of this. The fact that the hearing tests,

caloric tests and rotation tests are normal in most of these cases does not point to a peripheral lesion. Unless there is evidence of peripheral vestibular pathology and until the question is definitely settled as to the location of the lesion that causes positional nystagmus, the patient should be given the benefit of the doubt and be given a thorough neurological examination to search for any CNS pathology.

The following are a few remarks about my cases that may bring out some points and problems not already mentioned: my first patient, as previously mentioned, emphasized to me that repeated tests may be necessary to elicit positional nystagmus. This positive evidence of an organic basis for his dizziness, which had not been detected before, was very important in his disability rating. He was the first of five cases following a head injury. Disability, industrial medicine and medicolegal aspects are a big problem in these cases.

One patient was 70 years old and had no other pathology than her positional nystagmus. Her dizziness stopped within a month. Pyribenzamin did not help her dizziness.

Mrs. B., age 34, was in the U.S.N. Hospital for weeks under investigation. Her first dizzy attack came on during pregnancy several years before. She had had several series of attacks. No one had noted any nystagmus previously and she was beginning to wonder if she were imagining it. She had completely negative findings except the positional nystagmus, which could be produced very regularly whenever she moved from the erect to the supine position. It continued for a month. We took movies that were not good enough to show at this time. With the last series of attacks she had very severe occipital headaches. No treatment, including Dramamine, helped her dizziness, although it did help her nausea and vomiting. Dramamine did not help the dizziness in three other patients and made one worse.

Dr. Howard, an intern at Oak Knoll Hospital, had some dental work done with his head far extended and some heavy pounding on his teeth the morning of onset. His dizziness lasted about a month. He had one very remarkable observa-

tion: He is an excellent swimmer and during this time he noted that when he was swimming under water and made a turn he became dizzy and he had complete loss of his orientation as to whether he was swimming deeper or toward the surface. This scared him so that he requested the lifeguard to watch him when he swam under water. Such a loss of orientation might be disastrous to an aviator.

A Ford Motor employee had a fractured skull in July, 1948, and was under the care of very well known neurosurgeons who had found no organic reason for his dizziness when he changed the position of his head. Marked positional nystagmus was found, after several attempts, when he went from the erect to the supine position. The question has repeatedly come up as to his ability to work and his prognosis. At the present time he does not show positional nystagmus, but he does say that he is dizzy when he moves his head. Any patient who has postural vertigo and positional nystagmus is surely not able to work as a carpenter or a structural steel worker where falling might result and cause further injury. The prognosis of these patients is indefinite. Most cases seem to clear up in time, although some have recurrent attacks over a period of years. Apparently they do not have a progressive, degenerative process, although they may have recurrences.

Another industrial and medicolegal case had a severe head injury and had terrific nystagmus when he went from the sitting to the supine position. Following the caloric tests, which were normal, the patient did not have dizziness and nystagmus when tested and has had none since.

Mrs. G. had a slight spontaneous one degree rotatory nystagmus to the right. The caloric tests were essentially equal in both ears. When the patient went from the upright to the supine position the spontaneous nystagmus stopped for a few seconds and then became two degrees rotatory. Dramamine relieved the nausea but not the dizziness.

One patient who had positional nystagmus when seen once at home went to Lane Hospital for further observation. They reported a marked decrease in the caloric reactions on one

side but no other pathological findings. This is the only patient among my cases showing positional nystagmus who has had abnormal caloric tests.

Miss W. had dizziness for three weeks. After several attempts positional nystagmus was demonstrated several times, which was rotatory but with a strong vertical element. When we were taking movies the following day the camera broke down while she was having nystagmus and we were unable to produce further nystagmus. Dramamine upset her generally.

Mr. M., a U.C. student, had a head injury one year ago. His dizzy attacks then were similar to the present attacks of dizziness with a change of position. He had marked positional nystagmus. He also performed well at U.S.N. Hospital but the movie did not turn out well. He has not had an attack since.

Dr. F. at the Naval Hospital had a marked rotatory positional nystagmus when he put his head on his right shoulder. The following day when we took his movie we were unable to elicit the nystagmus in that position, but he did have an attack when he lay on his right side and this seems to indicate a rather strong vertical element in the nystagmus.

Mr. G., a professional baseball player, was hit with a baseball in front of his ear Sept. 7, 1949, and sent to me by an insurance company. He was stunned but not unconscious and was so dizzy he was taken to the hospital. The neurosurgeon reported no nystagmus when he lay on his right side, but when his head turned to the left there was a "very marked combined horizontal and vertical nystagmus with dizziness, nausea and vomiting. This subsided when he turned his head back to the right." There was no evidence of skull fracture nor of any other neurological pathology. He was in the hospital for three weeks and when dismissed no nystagmus was elicited. Another neurosurgeon saw him a little later and reported "nothing to suggest impairment of the central or peripheral nervous system," although the patient was having dizziness with a change of position. I first saw the patient Oct. 20, 1949. I was unable to produce nystagmus on several

attempts the first day, but the next day he had a violent nystagmus when he went from the erect to supine and with the head to left. The nystagmus seemed to be rotatory, vertical or diagonal. At another time it seemed to be rotatory at first and then vertical upwards; at another time it was vertical downward. On Nov. 7 he tried playing baseball. He had no trouble until he put his head back to catch a fly when he became violently dizzy and fell to the ground. When tested by having him put his head back from the erect to the optimum position and to the left he became violently dizzy and had a rotatory nystagmus to the left. Movies taken at this time were a failure but the present movies were taken a few days later.

This man had positional nystagmus develop in at least three different changes of position. A strong vertical element was present at all times and is shown clearly in the movie. He has complained of severe occipital headaches and a "black out feeling" during the severe attacks. The severe occipital headache has been a complaint of several other patients. A third neurosurgeon recently saw him and described his nystagmus as "typical rotatory in type and due to a disturbance in the labyrinth." There was no other neurological pathology. As his dizziness and nystagmus are so persistent I am wondering about his prognosis. He insists that he is going to play ball soon.

CONCLUSIONS.

1. Recurrent severe dizzy attacks that occur when the patient changes the position of the head are a common complaint.

2. Positional nystagmus is positive evidence of organic pathology causing the dizziness in some of these cases.

3. It is probably much more common than generally recognized.

4. Although the tests are very simple to perform, frequently it is necessary to repeat the test several times on two or three occasions in order to elicit the positional nystagmus which is very marked.

5. An otoneurological examination is not complete unless these tests have been performed on every patient who complains of dizziness that occurs with a change of position of the head or body.

6. Although it has not been definitely settled as to whether the cause of positional nystagmus is in the peripheral or central vestibular mechanism, it is the present conception that most of these cases are of central origin. This is brought out by the fact that the hearing tests, caloric tests and rotation tests are normal in many of these cases, as well as the fact that their nystagmus is frequently vertical in character.

7. Most of these patients get well spontaneously. No medication seems to effect the dizziness. Although the patients may have recurrent attacks of dizziness, the pathology apparently is not a progressive degenerative process, unless the pathology is due to a lesion such as a tumor, multiple sclerosis, etc.

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EXTERNAL OTITIS.

IV — Cytologic Study of Secretions.*†

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The present study was undertaken in an effort to understand the significance of the cytological components of secretions found in certain inflammatory diseases of the external auditory canal. It was thought that a study of the various normal and pathological cellular elements found in ear secretions might contribute to an understanding of the pathogenesis of external otitis and aid in the differential diagnosis of disease of the middle and external ear.

While bacteriological studies of secretions found in the external auditory canal have been numerous,[1-5] only an occasional reference has been made to the cellular components of the secretions. Politzer[6] examined the scales in acute diffuse external otitis and found large numbers of "micrococci." Proetz[7] examined the secretion following paracentesis in a case of allergic middle ear disease and noted cocci, a few leucocytes and epithelial cells, and an absence of eosinophiles. Recently Koch[8] reported an exhaustive study of the cytology of the secretions found in chronic otitis media and mastoiditis. He noted neutrophiles and bacteria in all discharges of chronic middle ear disease but did not report examinations of secretions of external otitis. Special emphasis was given to the presence of eosinophiles in allergic middle ear disease. Ts'en,[9] in 1926, studied the discharge from seven cases of acute and chronic middle ear infections. It was concluded

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that neutrophiles were present in both acute and chronic middle ear disease, but that in the chronic cases the neutrophiles were more apt to be degenerate forms. Dean and Pfingsten[10] while investigating the cholesterol content of various aural and nasal discharges also noted certain cytological features. They examined two cases of external otitis and noted epithelial cells and bacteria. Twenty-six cases of chronic otitis media and nine cases of acute otitis media were described, the discharges of which all contained neutrophiles.

Methods: The ear canals were cleansed of the mass of exudate by cotton swab or were irrigated with hypertonic saline,[11] and a smear was made of the exudate found in the sulcus of the canal. In cases of circumscribed external otitis the material was taken from the site of the discharge after spontaneous drainage or incision. The secretion was placed on the slide by rolling the applicator or very gently sweeping it across the slide so as to minimize morphological distortion of the cells. The smears were fixed by flaming and stained by a polychrome method (Wright's or two-stage eosin-methylene blue stain). In most instances a single smear was obtained prior to treatment. Some cases were followed by smears taken during the course of treatment in order to observe the cytological changes. A clinical diagnosis was recorded routinely prior to the inspection of the slide, although in a few cases the original diagnosis was revised in view of subsequent findings and the course of the disease.

The inflammatory diseases of the ear were divided into the following categories according to their clinical findings.[12] A small number of normals were included as a control.

A. Normal

B. External Otitis
1. Acute diffuse
2. Chronic diffuse
3. Circumscribed
4. Otomycosis
5. Seborrheic
6. Neurogenic

C. Otitis Media

D. Postoperative Mastoidectomy and Fenestration.

Smears obtained from ear discharge were examined for the presence of the following elements and the numbers of each of these were graded from zero to four plus:

Neutrophiles
Lymphocytes
Eosinophiles
Bacteria
Epithelial cells
Erythrocytes
Mucus strands*
Fungi.

Epithelial cells were classified according to shape, the presence or absence of a nucleus and the staining characteristics of the cell. The dispersion of the epithelial cells as singly, or in sheets or clumps was recorded. An effort was made to determine if there was bacterial invasion of the epithelial cells, but differentiation between true invasion of the cells and superimposed bacteria was difficult.

RESULTS:

Examination of the smears revealed certain general features. A nucleus was not visualized in more than half of the epithelial cells. It was found that no apparent significance could be attached to shapes of epithelial cells since in any single slide one would find a wide variety of shapes and arrangements of the cells. Eosinophiles were rarely found. Red blood cells were noted only in smears taken following a myringotomy or an incision of a circumscribed abscess.
The findings on smears in the various clinical categories as summarized in Table 1 are as follows:

*These were bluish or purplish staining filamentous structures varying markedly in size from 20 to 100 mu in length and 2 to 10 mu in width.

TABLE 1.

TOTAL CASES, 175. TOTAL SLIDES, 244.

Type	Neutrophiles	Lymphocytes	Strands	Bacteria	Epithelial
Normal 10 cases 11 slides	0	0	0	1	3-4
Acute Diffuse 53 cases 93 slides	0	0	0	3-4	3-4
Chronic Diffuse 32 cases 41 slides	0	0	0	3-4	3-4
Neurogenic 6 cases 10 slides	0	0	0	3-4	3-4
Seborrheic 21 cases 28 slides	0	0	0	2-4	3-4
Circumscribed 9 cases 9 slides	4	4	3	2	0-4
Otomycosis 4 cases 4 slides	0-4	0	0-3	0-2	0-3
Otitis Media 33 cases 40 slides	4	4	3	2	1-4
Postoperative 7 cases 8 slides	4	4	3	2	0-4

The figures indicate a grade of zero to four plus in the typical slide. Where variation occurred, the range is indicated.

Normal: Epithelial cells were seen in varying numbers. Bacteria were found in small numbers. Neutrophiles, lymphocytes or strands were not present in any of these slides.

Acute Diffuse: Epithelial cells were always present, usually in very large numbers. Myriads of bacteria were seen, although sometimes only two or three plus, the lesser numbers usually being found in cases nearing recovery. All cases seen early in the course of the disease or before treatment was instituted showed an absence of neutrophiles and lymphocytes. In seven cases followed by serial smears there were no leucocytes at the first examination, but neutrophiles and a few lymphocytes appeared later in the course of the disease. Strands were present in slightly less than one-third of the cases.

Chronic Diffuse: Epithelial cells were in profusion in almost every instance and myriads of bacteria were present in most cases. With one exception, leucocytes were never found. Strands were rarely seen.

Circumscribed: Eight of the nine cases revealed numerous neutrophiles and lymphocytes. Bacteria were usually present in small numbers. Strands were noted in eight of the nine cases and were absent in the same cases in which neutrophiles were absent. Epithelial cells were seen in variable numbers.

Otomycosis: An attempt to describe a typical picture would require more cases than were available in this series. One of the four slides showed neutrophiles but none showed lymphocytes. Strands were present in one smear, bacteria in three and epithelial cells in three.

Seborrheic: There were many epithelial cells and bacteria in almost all of the slides. Strands were rarely noted. No neutrophiles or lymphocytes were seen in 25 of 28 slides; the other three showed only an occasional leucocyte.

Neurogenic: Large numbers of epithelial cells and bacteria were present consistently. In nine of the 10 slides examined, no leucocytes were observed and the remaining slide showed two plus neutrophiles and one plus lymphocytes.

Otitis Media: Neutrophiles and lymphocytes in large numbers were found in all the smears. Strands were invariably present, usually in moderate number. Variable amounts of

bacteria were found. Epithelial cells were observed in small numbers except in chronic cases where they were more apt to be found in large numbers.

Postoperative Mastoidectomy and Fenestration: Epithelial cells, leucocytes, bacteria, and mucus strands were present in every case in varying amounts.

DISCUSSION.

This study was planned as an attempt to clarify the conglomerate group of inflammatory diseases of the external ear which are classified as external otitis without regard to the etiology, bacteriology or pathology involved. It has been possible to divide these cases into large categories on the basis of their clinical manifestations.[1,2] A more careful correlation with bacteriologic findings has contributed to a better understanding of some of these cases.[2] The cytologic study, it was thought, might serve to confirm, alter, or extend this classification. It might also provide an opportunity to check the clinical diagnosis by a simple office laboratory procedure; furthermore, while waiting for the culture report some more immediate and practical method for providing a clue to the causative agent was desirable.

This study, therefore, offers aid to diagnosis in two ways: *1.* An examination of the smear gives rapid and valuable information as to the probable causative agent, *e.g.*, bacilli, cocci, fungi, etc. *2.* The examination of the smear is helpful in distinguishing between purulent otitis media and external otitis (except the circumscribed type). The ear with a hidden tympanic perforation and a secondary diffuse external otitis may be difficult to differentiate from a diffuse external otitis with exudate covering the tympanic membrane. Fortunately, by cytological examination of the secretion, the differential diagnosis between otitis media with perforation and diffuse external otitis is usually clear-cut; the former shows neutrophiles, lymphocytes, mucus strands with a few bacteria and epithelial cells, while the latter presents a predominance of epithelial cells and bacteria, with perhaps an occasional leucocyte or mucus strand.

Perhaps the most striking finding in this study is the absence of neutrophiles in the secretions of acute and chronic diffuse external otitis. It is difficult to explain this observation unless the inflammatory reaction involves only the most superficial layers of the epidermis. The appearance of leucocytes in some cases might indicate that the superficial layers have been trespassed.

It is unfortunate that the cytologic findings at this time do not permit a differentiation of the various chronic categories of external otitis, *e.g.*, neurogenic, seborrheic, etc., but this is very much the condition in the pathology of these skin diseases, *viz.*, there are no clear-cut differential pathological changes.

CONCLUSIONS.

1. Cytologic examination of secretions in various categories of uncomplicated external otitis (except circumscribed) presents a uniform picture of epithelial cells, large numbers of bacteria and a striking absence of leucocytes.

2. The presence of neutrophiles, lymphocytes and mucus strands in secretions taken from the external auditory canal suggests a diagnosis of middle ear disease, circumscribed or complicated diffuse external otitis.

3. A cytologic examination of the ear secretions often will permit definitive diagnosis in cases where the differential diagnosis of otitis media or otitis externa is difficult.

4. The use of a polychrome stain technique as a rapid, simple office procedure for the examination of ear secretions is suggested as an aid in diagnosis and a guide for therapy.

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HEMORRHAGE IN EAR, NOSE AND THROAT.*

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A simple nose bleed does not cause us to worry, but all of us know there are very difficult hemorrhage cases in which we wished we knew one or two tricks to solve the problem. This discussion will bring out procedures, many of which have been passed on by men like you and, if they are new to you, will prove to be of value when the difficult hemorrhage case suddenly appears. Suffice it to say that almost every known cause of hemorrhage can appear in our field and almost every part is subject to hemorrhage. Knowing this, we should then be acquainted with the different means of controlling the hemorrhage. No attempt will be made to give a textbook coverage of the subject, such as giving the detailed blood anatomy and all the causes of hemorrhage in our field.

Epistaxis is most frequently encountered by us in the age group under 50 years. Each one has worked out a satisfactory method of stopping bleeding from the anterior part of the septum in children when the bleeding is brisk, but when there is just a little bleeding from both sides and a large area for a long period of time it presents a problem. Providing general causes have been eliminated, it can be controlled or cured in most cases by having the child wear a light plug of cotton in the nostrils for about 15 minutes at a time, two or three times a day. This follows Hilding's observations that shutting off the air in a nostril will allow the tissues to return to normal when the protective mucous sheath is present.

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Bleeding from the adenoid while the child is still in surgery may be controlled, other than by applying pressure from a string sponge, by applying the raw surface of a tonsil which has just been removed. It is first necessary to locate well the point of bleeding, then grasping the tonsil with an Allis clamp, press firmly over the bleeding point for about two minutes. This was first done on the basis of what most of us have done when a lateral sinus is bleeding, and the application of a piece of adjacent connective tissue, or better yet, a piece of muscle will stop the bleeding. The observation that vomitus tends to stop bleeding from the tonsil and adenoid area has suggested that dilute HCl be used. It is diluted down to about one-third strength, a sponge is soaked with it and pressure applied.

Bleeding from a hidden place in the nose in one under 50 years of age, and also to be found over 50 years of age, is often from the artery going to the inferior turbinate. A quick and efficient method of stopping the bleeding is to crush the turbinate over its entire length. It should be noted the the artery is at the surface on the lateral and inferior aspect just as the artery enters the turbinate at its posterior end, and it is at this point that the crushing does the work. The technique was suggested by a 30-year-old female patient who was quite exhausted after a week of attempting to stop the bleeding by the usual means, including postnasal and nasal packs, along with a number of transfusions. She suggested that a careful check on the blood supply be made and note which artery was nearest the surface. Her satisfaction was considerable when the technique about to be described stopped the bleeding. The nose is freed from clotted blood. An applicator with saturated cocaine solution is rubbed below and above the inferior turbinate from front to back. Novocaine is injected at the anterior end, at the mucocutaneous junction. A curved clamp without teeth at the end, and with blades long enough to include the entire length of the turbinate, is placed with the convexity outward along the attachment of the inferior turbinate. The clamp is closed tightly on the turbinate and left in position for a half minute before removal. If successful, the bleeding stops and the turbinate shrivels up,

due to its avascular state. No packing is necessary, no discomfort follows and in a week the turbinate has resumed its former appearance. Do not turn the turbinate upward or the artery will be torn from its moorings and the bleeding continues. Quite a number of hemorrhages in people over 50 years of age come from this source, and this method is quite satisfactory in that it can be done easily in the home.

If bleeding is not controlled, then a 5 cc. Foley catheter is inflated with water and used as a postnasal plug. If fortunate enough to make contact on the bleeding artery, it is entirely satisfactory and quite comfortable. The usual methods of gauze packing, both nasal and postnasal, are known to all, so will be passed over.

When bleeding continues, especially in one over 50, the electric cautery is used. The blood usually comes from the sphenopalatine artery near the posterior end of the middle turbinate. The patient is put in the hospital, given an intravenous pentathol anesthesia while in the head low position. Sitting at the head makes it easy for the operator. The palate is elevated by two catheters. A metal suction tip covered with rubber is passed through the nose and is guided by viewing through a large laryngeal mirror. It may be necessary to bend the end of the suction tip to make contact with the bleeding point. If good contact is made, the bleeding stops because it is all aspirated into the tube. The electric current is turned on and the tissues coagulated. The "cutting" and not the "coagulating" current is used because the "cutting" current is more superficial, thus resulting in less sloughing. The amount of current must be determined by each operator at the time. A note should be made in regard to the suction tube. Use a rubber tube small enough so that it will cling tightly to the metal tube, leaving only about an eighth inch of exposed tip. By first putting a few drops of oil in the rubber tube it will easily slide on the metal tube.

Considerable trouble in getting down to the external carotid artery has at times proved embarrassing, not only to some of us but to general surgeons when it was hoped he could do it

more efficiently. Following Dr. Barnhill's technique of blunt dissection and practicing on monkeys, it was found there was little difficulty in quickly exposing any desired vessel in the carotid sheath area. To review quickly, a skin incision is made just anterior to the sternomastoid, the fascia is nicked and the fascial layers are separated by inserting two Kelly hemostats, and gripping them firmly, the tissues are separated for the length of the incision. Use little or no retraction as the landmarks become distorted. It may be necessary to again nick the fascia before inserting the hemostats for another separation. By this blunt dissection, there is little or no bleeding.

Once in a while it is necessary to ligate the ethmoid arteries. This is done by the external route, and upon exposing them, instead of ligation the use of the "cutting" current works well. This was thought to be a procedure without danger until one of our members had a case in which blindness followed the cauterization of the posterior ethmoid artery. If heat from the cautery was the cause, then ligation is to be preferred.

Bleeding from the ear has been encountered mostly from blebs. When blebs are seen before they have ruptured, a quick way to drain them is to instill 25 per cent magnesium sulphase in glycerine. It is quite painful, so the patient is given something for pain before the treatment. In a few minutes the blebs either rupture or flatten out. The magnesium sulphate dissolves with difficulty. A warmed mortar and pestle (about 110°) is used and the magnesium sulphate is ground into the glycerine over a period of about two hours. Only occasional grinding is necessary.

Bleeding from the lungs or esophagus is of interest only to the endoscopist and will be passed over lightly. A good internist or chest man is quite necessary to give aid in proper diagnosis and treatment. Seldom is a bleeding point found on bronchoscopy as the bleeding comes from farther down. A technique has been described for plugging a bronchus by heavy cautery, and in this way the bleeding is dammed up in

the lung below. Bleeding from the esophagus may require treatment through the esophagoscope or the aid of a surgeon may be necessary. No personal experience of value can be passed on to you.

Recently Dicumarol has been used in treating multiple sclerosis, and there is constant danger of lowering the prothrombin time to the point of spontaneous hemorrhage throughout the body. Such a case was recently encountered and since other cases are likely to occur, it is well to relate our experience. There was an emergency call to do a tracheotomy on an almost unconscious 30-year-old female who was on a maintenance dose of 50 mg. of Dicumarol. Examination showed a submucosal hematoma of the posterior pharyngeal wall, so large that it pressed against the tongue. By the time the patient arrived in surgery breathing had stopped and a quick tracheotomy was done, after having injected novocaine with adrenalin. In the meantime the prothrombin time was found to be zero. A blood transfusion had been ordered. Within about 10 minutes after the tracheotomy was done hemorrhaging began so profusely that it could not be aspirated fast enough. The patient was turned over for postural drainage and the transfusion was started. Finally two blood and one plasma transfusions were going in three different veins. After about an hour the bleeding stopped and a blood pressure could be obtained for the first time since surgery. The systolic was 40. Looking back, there seems to be little else that could have been done, but this serves as a warning to us that we have many potential severe hemorrhage cases in those patients who are going along on maintenance doses of Dicumarol.

Many of you have in mind other methods of controlling hemorrhage, such as the use of Gelfoam and similar preparations; salt fat pork, as told to me first by Dr. Arthur Jones; crisscross incision and Sylnasol injection for bleeding from the anterior part of the septum as experienced by Dr. J. Mackenzie Brown; the use of sea sponges as described by Dr. Gilbert Roberts, in which they are used not only for splinting but for controlled bleeding as well; injection of novocaine with

adrenalin in the tonsil bed for any stage of post-tonsillectomy bleeding as described to the author by Robert Odom, and many others could be related.

It is hoped there will be a free exchange of ideas so that we may all profit by the other man's experience. We should make an effort to know well the methods of controlling our most difficult hemorrhage problems and there will certainly be less apprehension and time wasted when the emergency arises.

THE MANAGEMENT OF NASAL FRACTURES.*

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Nasal fractures, like tonsils, apparently have come to be regarded by the otorhinolaryngologist as a subject too elementary to warrant further study or discussion.

A review of the literature for the past 10 years appearing in the American journals devoted to otorhinolaryngology revealed a voluminous bibliography on rhinoplasty but only a half dozen noteworthy articles on the management of nasal fractures.[1-6]

Even the standard textbooks on otorhinolaryngology have passed hurriedly over this subject with few or no details as to technique.

With the rhinologist entering the field of rhinoplasty, it seems strange that one of the basic causes of the need for rhinoplasty, as well as for submucous resection, should receive so little attention.

It would appear that the rhinologist, interested in doing a better job of setting a broken nose, will have to refer to texts on plastic surgery and rhinoplasty in order to find the answer.[7,8]

It is unfortunate but true that nasal fractures all too frequently are dealt with hurriedly and inadequately, leaving the patient a result far from satisfactory cosmetically and, more important, functionally. In fact, many patients are told at the time that a corrective operation will be necessary later.

It is the purpose of this presentation to emphasize the need for a clear understanding of the anatomy of the external nose,

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particularly the relationship between the bony arch and the cartilaginous structures and the importance of the latter in the end-result of nasal trauma.

Second, a technique permitting deliberate manipulation under adequate anesthesia, followed by adequate splinting, inside and out, will be described.

The framework of the nose is composed of a bony and a cartilaginous portion. The former is made up of the paired nasal bones, fused together in the midline, and articulating with the nasal process of the frontal bones superiorly, and the maxilla laterally. The strongest portion of this bony arch is at its attachment to the frontal bone, while its weakest and most frequently fractured portions are the free margins of the nasal bones inferiorly, and their attachments to the maxilla laterally; however, the bony arch is only half the picture, although all too frequently it is all that receives attention during hurried reduction.

The upper lateral cartilages, fused to the dorsal border of the septum, are loosely attached to the lower border of the nasal bones by means of fibrous tissue. Their intimate connection with the septum results in a guy rope action, with the septum acting as a ridgepole. Any forcible displacement of the septum may be followed by a tear in their fibrous attachment, with a lengthening of one guy rope and a shortening of the other, and a resulting deformity of the cartilaginous dorsum.

The septal cartilage, which rests in a groove in the vomer along the floor of the nose, has a doubly important rôle. It divides the nose into two air chambers, and anything that changes its position will alter the breathing function. Second. as mentioned previously, it serves as a ridgepole, guyed by the upper lateral cartilages.

The displacement of the septal cartilage from its groove in the vomer is a common occurrence; it has been shown to result from trauma at birth and from frequent falls and blows during childhood. The resulting deformity produces

nasal obstruction, drooping of the tip and sagging and angulation of the cartilaginous dorsum. If treated early before fibrous union takes place, it can readily be corrected with thumb and forefinger, or with an Asch or Walsham septal forceps under light ethyl chloride or Vinethene anesthesia. If the bony dorsum has not been fractured, and it frequently is not, this is usually all that is necessary.

Injuries in adolescent and adult life, however, present a problem that requires a carefully planned and worked out technique. First of all, the rhinologist should not allow himself to be rushed into a hurried, spur-of-the-moment attempt at reduction under unfavorable circumstances.

We found from a large number of cases in the Navy that reduction could be delayed up to a week; and in cases of massive swelling and hematoma it was far better to wait until we could see what we were dealing with. X-ray films were not particularly helpful except in cases of depressed marginal fracture, which a soft tissue lateral view will show in the absence of clinical findings.

Lateral deviations and dorsal depressions should be looked for carefully, remembering that only the cartilaginous dorsum may be disturbed. If so, crepitus may be absent and point tenderness slight. Careful palpation may reveal a depressed fracture on one side only, at the junction of the nasal bone with the frontal process of the maxilla, without any apparent deformity because the dorsum is not shifted and the swelling hides the depression. Fractures of this type may produce a most surprising and disturbing deformity when the swelling subsides.

Finally, the nasal mucosa should be thoroughly shrunken with a spray of 10 per cent cocaine and adrenalin, all clots and secretions removed with spot suction, and the septum carefully inspected. Misalignment may be due to an old deformity or the result of the present injury. The presence of hematoma would suggest the latter; also, the septum may be found to be movable after intranasal anesthesia has been accomplished by sphenopalatine and anterior nasal nerve block with cocaine applicators.

If it has been determined that reduction is necessary, external anesthesia is accomplished by 2 per cent procaine nerve block as follows:

One centimeter above the inner canthus on each side a small wheal is made with a 26 gauge needle. Then a 22 gauge needle is inserted along the medial orbital wall to a depth of 2 cm. and 1 cc. of solution is injected. This anesthetizes the supra- and infratrochlear nerves which supply the upper part of the nose and also the anterior ethmoid nerves which supply the anterior superior portion of the septum. The infraorbital nerves are blocked by similar wheals 1 cm. lateral to the ala nasi, followed by injections into the infraorbital foramina. This anesthetizes the lower part of the external nose. This technique avoids infiltrating the tissues overlying the framework of the nose, and preserves an undistorted field.

Only two instruments are necessary for the ensuing manipulations: a narrow Lagenbeck periosteal elevator with a slightly curved tip is used to elevate depressed fragments; a Walsham septal forceps, which is similar to an Asch forceps, but with wider blades which cannot be completely closed, is the ideal instrument to rotate and unlock fragments and to realign the septum. One must learn to visualize mentally the position of the fragments and to note whether or not they are locked in order to realign them without undue force or further comminution; however, realignment of the septum may require considerable leverage, and frequently a lateral displacement of the dorsum which has not responded to manipulation of the bony arch will suddenly snap into alignment as the septum is straightened. A depression of the cartilaginous dorsum also will be corrected by elevation of the septum back into its groove in the vomer.

All these manipulations can be carried out unhurriedly and carefully with a minimum of bleeding and discomfort under the described anesthesia. One may take as long as 30 minutes to accomplish a desired result without undue complaint from the patient. Premedication with a hypnotic barbiturate is desirable, but morphine or a similar narcotic is rarely necessary.

After the manipulations have produced a satisfactory result cosmetically and functionally, the nose is splinted inside with foam rubber sponge material. This can be obtained at any 10 cent store in the form of powder puffs which can be sterilized by boiling. Cut in two, they require very little additional shaping to fit high in the nasal vault. Rolled or folded, they are inserted through the anterior nares, and they promptly spring back into shape, filling the nasal cavity much more satisfactorily than a Simpson splint. They exert a smooth, even pressure and stay in place without anchoring. It is the author's belief that they reduce intranasal swelling and hematoma materially and that they soak up secretions "like a sponge."

The external splint prevents swelling and hematoma, and holds bony fragments and torn cartilages in place until healing is well along. Soft lead or copper splints are advocated

by many otorhinolaryngologists, while the rhinoplastic surgeons prefer dental stent; however, believing the former too difficult to shape and hold in place and the latter too heavy and cumbersome, the author finally decided to emulate the orthopedists and has been using a plaster dressing with excellent results. Before the plaster is applied, the eyes are protected with a few drops of mineral oil or olive oil. Then a cotton flannel butterfly is shaped to the nose and forehead and small strips of J & J Specialist Plaster, lightly soaked, are applied layer on layer, longitudinally and transversely, until a sufficiently heavy spica is built up. This is allowed to dry, which it does rather quickly, and then strapped snugly in place with adhesive strips across the forehead and cheeks.

The sponges are left in place five days, the patient receiving procaine-penicillin daily, and the plaster dressing is removed after 10 days.

In the author's experience the improved results of the above technique have more than made up for the extra effort involved, and while perhaps not as remunerative as submucous resection or rhinoplasty, have been a source of great personal satisfaction.

SUMMARY.

Nasal fractures all too frequently are dealt with hurriedly and inadequately, leaving an unsatisfactory cosmetic and functional result.

A clear understanding of the anatomy of the external nose, particularly the relationship between the bony nasal arch and the cartilaginous structures, is essential to the successful management of nasal fractures.

A technique permitting deliberate manipulation under adequate anesthesia, followed by adequate splinting, inside and out, is described.

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REHABILITATION OF THE PRESCHOOL DEAF CHILD.*†

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The purpose of this paper is to present the experience of a modest clinical research program, a program designed for the rehabilitation of the preschool deaf child in a metropolitan area where virtually no other facilities exist. It is with reference to such areas of limited resources that I direct my remarks to you as otologists, rather than to teachers of the deaf or to those already participating in more complete programs.

In 1944, Dr. R. P. Guilder[1] reported preliminary results of an experimental program which offered intensive remedial assistance to a small, select group. This initial work was of such significance as to demonstrate the need for an expanded program. Dr. Schall and Dr. Meltzer, cognizant of the need for a closer alliance between the surgical and nonmedical therapy of deafness, gave us the opportunity of continuing and expanding this program. It is now an integral part of our plan for resident training at the Massachusetts Eye and Ear Infirmary, concomitant with the study of otosclerosis and fenestration technique. Its effectiveness is based upon the six following principals:

1. Diagnosis.
2. Estimate of Hearing Capacity.
3. Parent Education.
4. *Early* Application of Remedial Measures.
5. Continuous and Sympathetic Follow-Up Program.
6. Flexibility of Program as Needed.

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†From the Winthrop Foundation at the Massachusetts Eye and Ear Infirmary.

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DIAGNOSIS.

The diagnosis of deafness in this age group may be confused with any of the conditions which cause a failure of language development. They are motor speech delay, developmental word deafness, mental retardation, lack of motivation and emotional traumata.

The following developmental language disabilities are an amorphous group, familial in tendency, associated with a disturbance of dominance in handedness and lacking in organic brain pathology.[2]

1. Developmental Word Deafness.
2. Motor Speech Delay.
3. Specific Reading Disability (Alexia).
4. Special Writing Disability (Agraphia).
5. Stuttering.
6. Developmental Apraxia (Clumsiness).

Stuttering, apraxia, and reading and writing disabilities present no problem as regards the differential diagnosis of deafness. They are frequently present, however, in combination with one of the other syndromes more easily mistaken for deafness. Motor speech delay is frequently seen to coincide with efforts to train a child away from a preferred left-handedness. Children with this disorder are attentive to and understand the spoken word. Most of them eventually develop speech to a varying degree of success. With their ability to understand speech they appear intelligent and adaptable.

Developmental word deafness, which would coincide with a pure sensory aphasia of the adult, is lack of understanding of the heard word. This interference of the receptive mechanism causes a delay and a distortion of speech, and prevents the acquisition of all these concepts formulated by the spoken word. The deaf child somehow manages to acquire these verbal concepts and subsequently his retardation is not so severe. The child with developmental word deafness is, at first, completely inattentive to speech and his early environmental investigation is limited to the medium of vision and

touch. Gradually he begins to "echo" monosyllables, and with increasing maturity he learns the meaning of single words and may acquire an appreciation of very simple sentence structure with, however, errors in both pronunciation and grammar. Thus from the standpoint of symptomatology and therapy, he is an aphasic; but he is not classified as such because of his positive family deviation in handedness and absence of organic brain pathology.

The child with borderline intelligence, whose appearance may pass for normal and who may not bear the obvious stigmata of retardation, is frequently believed to be deaf, primarily because of his poor language behavior; so again his performance in nonverbal tests becomes important. The most constant finding to me in establishing a tentative diagnosis of retardation is the presence of what might be called "visual inattentiveness." The deaf child with his normal personal-social behavior is interested in people. He watches people, their movements, and is sensitive to facial expressions and often attentive. The retarded child may have a compulsive interest in objects, but is emotionally and intellectually unresponsive to people. He is prone to show poor muscular coordination, manual clumsiness and stereotyped purposeless movements.

Emotional disturbances, particularly those due to parental overprotection, are causative factors in speech delay. The child of oversolicitous parents, with his needs constantly anticipated, will lack the incentive for developing speech. At the other extreme, the institutionalized child without the give and take of ordinary home life develops a poverty of self-expression.

Etiology. A knowledge of the etiological factor responsible for the deafness may be helpful in estimating the hearing capacity and in predicting a tendency toward progression. In the order of frequency of occurrence in these patients, I have classified the deafness as congenital, infectious, hereditary and traumatic. The term congenital has been restricted to mean those who were born with defective hearing without any

evidence of birth trauma or significant family history. Inherited deafness, present at birth, has been classed as hereditary rather than congenital. The child with a strong family history of inherited deafness is faced with the statistical probability of a further loss of hearing (see Fig. 1). I have not

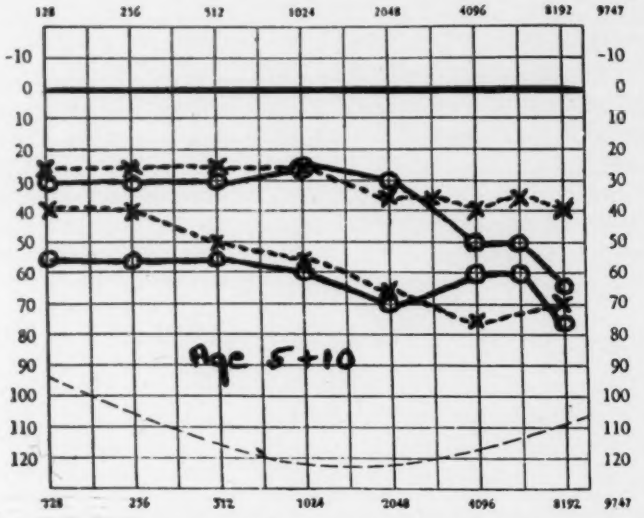

Fig. 1. Audiograms of a child taken at ages five and 10. She has a strong history of inherited deafness.

noted this retrogressive tendency in the congenitally deaf group where the deafness was the result of maternal rubella or erythroblastosis fetalis; furthermore, the rubella cases have almost invariably shown a correlation between the severity of the hearing loss and the month of maternal infection. Rubella occurring in the first month of pregnancy has resulted in a profound deafness in the offspring, while the deafness resulting from infection in the third month of pregnancy has not been so severe.

PARENTAL EDUCATION.

1. Personal discussion with physician.
2. Reading material.
3. Group conferences, lectures and social activities.
4. Follow-up interviews with social service.
5. Correspondence courses and instructive manuals.
6. Attendance at classes for speech and auditory training.

The medical consultations should clarify in the minds of parents the nature and degree of their child's hearing loss and should further emphasize the futility of a circuitous search for curative treatment which all too often results in such ill-considered procedures as prolonged vitamin therapy, radium treatments and unwarranted attacks on the nasopharynx. Parents of defective children tend to react with feelings of guilt and personal inadequacy. These feelings must be discussed and resolved if we are to secure for the child an atmosphere free from tension and overcompensation. Assistance should be given in understanding the limitations of a hearing aid and the exacting visual discrimination necessary for the child in speech reading.

Specific reading sources are the *Volta Review*, "Hearing and Deafness" by Hallowell Davis, "Developmental Diagnosis" by Gesell and Armatruda, "Opportunity and the Deaf Child" by Ewing and Ewing, and *Hearing News*, published by the American Hearing Society. If the child is in the two to three-year age group and has had no constructive guidance, his parents may enroll in the John Tracy Correspondence Course. A child who has had this instruction, when he comes to me, is a year ahead of the same aged child who has not been so instructed. The parents of our children are given detailed advice in the development of the "conditioned auditory response," and the parents themselves then become participants in a constructive program. With a play approach as a background, the child is taught to take his turn in some simple game, in response to a sound stimulus. This is the basis for ensuing audiometric and speech evaluation.

Group association with other parents of deaf children has proven helpful. At the Winthrop Foundation the parents spontaneously organized a group which meets four to six times a year and sponsors educational programs and social activities. Once a year the Winthrop Foundation organizes a group conference designed specifically for the public school teachers of our handicapped children. Here an attempt is made to include those teachers as an integral part of the remedial program and as an essential unit of our clinical research study.

Whenever possible the mother is urged to be present for her child's lessons in acoustic training, speech reading and speech development. In no other way can she develop such a keen appreciation of this formidable problem, and with expert guidance the mother gains insight and skills enabling her to continue with her child at home.

ESTIMATION OF RESIDUAL HEARING.

1. History.
2. Spontaneous Response (blinking, jumping, turning, locating).
3. Learned or Conditioned Response (gross sounds, voice, audiometer).
4. Observation of Child's Basic Language Ability.
5. Psychometric Evaluation.
6. Vestibular Function Tests.
7. Galvanic Skin Resistance Method.

The subject of hearing tests habitually calls to mind the desire for an audiogram. We are concerned here with the child's potential capacity both to hear speech and manipulate language. It is obvious that the information derived from an audiogram is limited. It tells us, at best, his threshold of awareness for several isolated frequencies of the sound spectrum. It tells us little as to his auditory performance above threshold and nothing of his discriminatory capacity upon which the hearing of speech depends. We then call upon a combination of resources not commonly used in the testing of adults.

First is the history. I have learned not to underestimate the information which may be obtained from the parents. "What do you as the mother think your child hears? (We disregard the handclap, airplane, etc.) What evidence can you recall that he has heard voice sounds? Will he attempt to repeat simple words spoken close to his ear? Will he turn to his name in a loud or moderate voice at 20 feet? Does he have any understanding of single words or phrases through hearing alone?"

Second, What have been his attempts at vocalization? Does he have the urge to talk? Does he attempt simple words to express his desires? Does he have the nucleus of a vocabulary learned through hearing alone?

The child's reflex response to sound stimuli is inconsistent, uncertain and always well above threshold. The infant under a year will blink; between one and two years he will add to his response, muscular jerks; shortly thereafter his head will turn in an attempt to locate the source of the sounds. To me, his spontaneous response has limited value, but it prepares the way for his development of a "conditioned auditory response." I use a percussion instrument, a cowbell, a police whistle and a set of pitchpipes whose range is from about 200 to 1,800 cycles per second.

The most single reliable method has been the learned or conditioned auditory response. While sitting at a small table, the child is taught to take his turn at some simple game (push a penny into the circle, a marble in the milk bottle, or put a brightly colored car into the toy garage) and he is taught to take his turn only at the command of a sound. At the first visit this can be demonstrated to the parents, who can then perfect the conditioning at home. With this method he is then tested with gross sounds as mentioned. From there we proceed to the use of voice sounds, first the vowels (either nonsense syllables or words). We are especially interested in knowing whether he can hear the consonants and he is tested with the plosives, the voiced and unvoiced consonants, and the diphthongs.

If he hears consonants with the unaided ear, he is no more than partially deaf. If he hears only an occasional vowel, loud and close to his ear, he is profoundly deaf. The next step is to try the earphones of the audiometer. I use first the frequency of 500 c.p.s. at high intensity to condition him to the technique. He is next tested at 2,000 c.p.s. since we are particularly interested in his hearing at this frequency. The majority of these children have descending curves (as related to decibels rather than sound pressure level) and if there is hearing at 2,000 c.p.s there will probably be hearing at 1,000 c.p.s. and 500 c.p.s. An elaboration of this method is the "peep show" technique advocated by Dix and Hallpike,[3] wherein there is a greater reward factor for a correct response. The child, using earphones, looks into a box of colored pictures. In order to change the picture he must pull the treadle while the pure tone stimulus is on. In this way his threshold may be approached for the various frequencies.

During the entire procedure we are observing the child's response to speech sounds, his ability to imitate, his desire to express himself.

The psychometric evaluation is desirable when facilities are available. The test is performed on a nonlanguage basis, and the more superior the ability of the child the more optimistic we may be regarding his eventual rehabilitation.

I have been impressed with the rather meager amount of additional information derived from vestibular tests. Irrigation of the child's ears with ice water effectively shatters his confidence in the doctor, who then may have difficulty in regaining his cooperation. By and large, if one is unable to elicit a vestibular reaction, the ear is profoundly deaf. If there is a significant response of the static labyrinth, he may be only partially deaf.

The galvanic-skin resistance method, as reported by Bordley, Hardy and Richter,[4] depends upon conditioning the child to a shock stimulus preceded by a pure tone. After he has been conditioned his sweat glands will respond to the pure tone alone, without the shock. This measurable response assumes

a "normally reacting sympathetic nervous system." In short, it is an ingenious but time consuming method, requiring a team of trained personnel, and as yet it is not adaptable to mass testing.

REMEDIAL MEASURES.

Specific remedial measures embrace a combined program of acoustic training, speech-reading and language development. If the child has a residuum of hearing amenable to re-education and his psychometric evaluation is favorable, he is given a hearing aid. He is not "fitted" to an aid since he lacks the capacity to judge the sensations of comfort, loudness, clarity and quality. The factors considered in the selection of an aid are its cost, its past performance and its amplification characteristics as published by the American Council of Physical Medicine. The congeniality of the local hearing aid representative is also worthy of consideration. If the patient is partially or profoundly deaf, the better ear is used since binaural hearing is seldom possible. Our experience with the more powerful group hearing aids, such as the Maico and Warren Training Units has been limited.

Fig. 2 shows the remedial needs of the child in relation to his hearing capacity as a function of the audiogram. The partially deaf child should invariably have a hearing aid and lip reading regardless of whether he is able to obtain acoustic training and speech lessons. His proficiency in speech reading must not be allowed to develop to such a degree as to subordinate the utilization of his residual hearing. Recommendations for a hearing aid for the profoundly deaf child, however, should be made with caution, particularly if its procurement cannot be followed by expert guidance in speech therapy and auditory training. The profoundly deaf child needs all the speech reading proficiency he can acquire as his residual hearing can be exploited to a lesser degree.

We have at the Winthrop Foundation three well trained teachers of the deaf whom we employ on a half time basis in the hospital. The child is accompanied by his mother and has one, two or three hourly lessons weekly. Hearing is used inso-

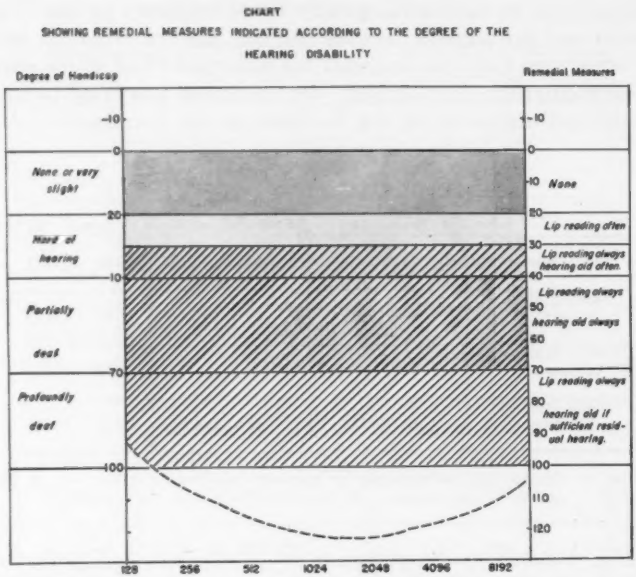

Fig. 2. Shows the remedial needs of the preschool deaf child in relation to his hearing as a function of the audiogram. The partially and profoundly deaf child also needs instruction in speech development.

far as possible, supplemented by the tactile, visual and kinesthestic senses. Since the teaching aspect of this program has not been subsidized, parents must pay for the instruction periods. In this respect it is significant that the State Department of Special Education offers no help, financial or educational, to the child in the preschool age group. We have been assisted in help with speech training and lip reading by both the Sarah Fuller Foundation and the Boston Guild for the Hard of Hearing. The Boston Guild has gradually lowered the limit for the children assisted to the five to six-year level, thus approximating the preschool age group. They offer group lessons once weekly. The Sarah Fuller Foundation, by sending its teachers directly to the home once weekly, offers valuable assistance without charge but, like the Guild, needs the services of a medical and diagnostic center where recommendations can be made. In Massachusetts we have had the

cooperation of such strategically located centers as the Worcester and Springfield Leagues for the Hard of Hearing, both of which are now able to assist the preschool child in his early speech-language development. Our program has tried to integrate and utilize all of the facilities at our command.

ADEQUATE SOCIAL SERVICE FOLLOW-UP

	Known 160	Unknown 157	Total 317
Hearing Aids Recommended	73	53	126 (39%)
Hearing Aids Obtained	57 (79%)	36 (66%)	93 (74%)
Speech Training Recommended	90	78	168 (58%)
Speech Training Received	65 (72%)	46 (56%)	111 (65%)

Fig. 3. Shows the percentages of recommendations carried out in two groups of children, one known and one unknown to social service.

Fig. 3 shows the importance of an adequate social service follow-up program. Seventy-four per cent of the hearing aids recommended were purchased and 65 per cent of the patients, for whom speech training was recommended, obtained it. The percentages on all recommendations carried out were higher by 13 to 16 per cent in the group known to social service. The fact that more than one-third of the children did not receive the recommended speech training reflects the acute shortage of available trained teachers for the deaf.

Now, you ask, what results have you had with this program . . . and by what criteria do you measure success? The ultimate criterion is that of school placement. Fig. 4 shows the results of a group of 55 children, trained by us, and followed for a sufficient length of time so that their fate, as regards school placement, is reasonably certain. Of 38 partially deaf children, whose average loss was close to 70 db, 35 are in public or private schools. Only three are in a school for the deaf. Of 17 profoundly deaf children, eight are in public school.

GROUP HAVING TRAINING AT HOSPITAL

	Partially Deaf 38	Profoundly Deaf 17
Public School	29 (76.3%)	7 (41.3%)
Private School	6 (15.7%)	1 (5.8%)
Deaf School	3 (8%)	9 (52.9%)

Fig. 4. School placement of a group of 55 children who had intensive, long term speech therapy under medical supervision.

Fig. 5 shows the school placement of another group, advised and studied by the Winthrop Foundation, but receiving sporadic and somewhat inadequate speech-hearing training elsewhere. Only 49 per cent of the partially deaf and 1 per cent of the profoundly deaf children in this group were able to receive public school instruction.

Inadequate Teaching

School	Partially Deaf (59)	Profoundly Deaf (75)
Public	29 (49.1%)	1 (1.3%)
Private	16 (27.1%)	1 (1.3%)
Deaf	14 (23.8%)	73 (97.4%)

Fig. 5. School placement of a group of 134 children whose instruction in speech development was inadequate.

SUMMARY.

Facilities for the rehabilitation of the preschool deaf child in this country are inadequate. The majority of partially deaf and many of the profoundly deaf children can be rehabilitated, through early intensive remedial measures, to the level of public school placement. The enthusiasm, prestige and knowledge of the otologist are essential for the development of programs to meet the needs of these crippled children.

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ABERRANT SALIVARY GLAND TISSUE AT THE BASE OF THE TONGUE.*

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In a recent article, O'Neil[1] has reviewed all the cases reported in the literature on aberrant salivary gland tissue found in the tonsillar fossa region. Considering the thousands of tonsillectomies performed each year and the proximity of the salivary glands to the tonsillar area, it is surprising that only seven cases of aberrant salivary gland tissue have been reported in or about this region.

We have found infrequent references to aberrant salivary gland tissue in such other locations as the lip, palate, gums and in the vicinity of the pituitary. We have found no reference to aberrant salivary gland tissue associated with other developmental abnormalities such as were present in the case we are reporting.

REPORT OF CASE.

This seven-year-old child was first seen at the ENT clinic on Jan. 20, 1949. The main complaint was congenital microtia of the right ear. He had only a very limited vocabulary and a pronounced speech defect which his father attributed to imperfect hearing. The child had three older siblings who were normal both mentally and physically, and this was cited by the father as evidence that the child's backwardness was wholly the result of imperfect hearing.

A further complaint was that the boy was a continuous mouth breather. He had had a tonsillectomy and adenoidectomy at four years of age. The physician had told the father at the time of the operation that he was

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unable to find any tonsil in the right fossa and that the condition was, therefore, a congenital deficiency. The father had deducted from this conversation that the doctor may have also missed the adenoid and that this could be the cause of the mouth breathing.

General physical examination demonstrated a well proportioned child of good nutrition. He was able to hear conversational voice but usually declined to answer questions. When he spoke it was usually monosyllabic and there was a definite speech defect. He seemed shy and was essentially a mute. He appeared to be slightly substandard mentally.

There was a congenital microtia of the right ear, there being no meatus and only a remnant of a lobule as shown in Fig. 1. The left ear was undersize and somewhat deformed as shown in Fig. 2. The external auditory canal on this side was partially stenosed. The audiogram for the left side was only slightly lowered for air and bone conduction and for the right side there was no air conduction and only questionable bone conduction. Mastoid Roentgenograms showed diploic cells bilaterally, but there was less development of the mastoid process on the right. It was the opinion of the Roentgenologist that the right internal ear and middle ear were at least partially developed.

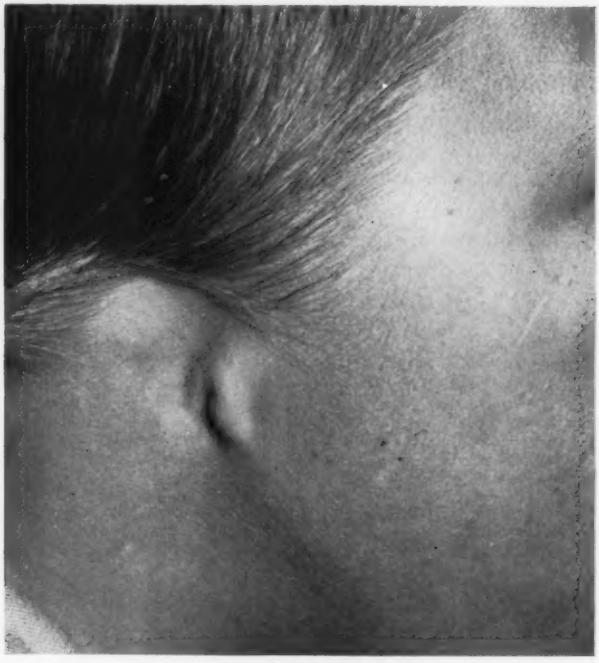

Fig. 1. Microtia of right ear.

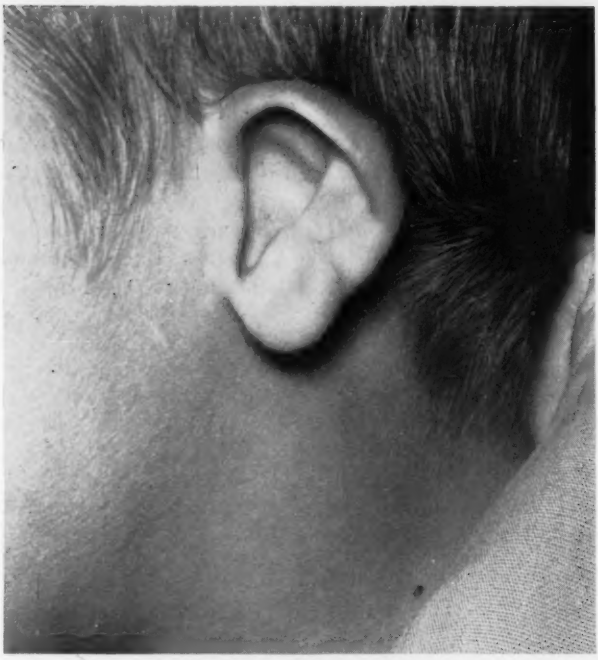

Fig. 2. Maldevelopment of left pinna.

Oral examination showed a congenital absence of the right faucial pillars and maldevelopment on the same side of the soft palate. The patient was able to blow up a balloon, to blow out his cheeks and to swallow without trouble, indicating that the muscles of the tongue, palate and pharynx were competent. The palate was highly arched, of the cathedral vault type. There was a moderate amount of pharyngeal lymphoid tissue palpated in the adenoid tonsil area. On attempting indirect laryngoscopy, a mass of tissue about the size of an unshelled almond was seen attached to the base of the tongue and below the right tonsillar fossa. At the time this was presumed to be an aberrant tonsil. This seemed most likely in view of the fact that no tonsil was present either at the time of the examination or reportedly at the time of the original tonsillectomy and adenoidectomy.

Roentgenograms of the chest showed the heart to be within normal limits of size and shape and the lungs clear. There was a synostosis between the anterior portions of the first and second ribs on the left.

The routine laboratory findings were within normal limits.

Because of the apparent difficulty with breathing and because of the speech defect it was decided that removal of the tissue at the base of

the tongue might be beneficial. The operation was performed on March 2, 1949, with a tonsil snare. The tissue cut similar to fibrous tissue, and there was scarcely any bleeding, a condition identical to that reported by Hitschler and Cope.[2] The tissue was rather pale in appearance and of about the same firmness as that of a salivary gland.

The pathological report follows: "*Gross:* There is a 2.5 x 1 cm. mass covered by heavy verucous mucous membrane except for a small portion which is rough and fibrous. *Microscopic:* (See Fig. 3.) A section of stratified squamous epithelium underlaid by mixed type salivary gland tissue and one large duct. No lymphoid tissue is visible. There is considerable fibromuscular tissue around this mass."

Fig. 3. Twenty times magnified histological section of salivary gland tissue.

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REACTIVE SYMPTOMS AFTER THE INTRATHECAL ADMINISTRATION OF PENICILLIN. (MENINGEAL PSEUDOEXACERBATIONS.)*

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It is a common observation that patients who are convalescing from meningitis following penicillin therapy may suffer from headache, nausea, vomiting and fever. We formerly felt that in such cases the symptoms were due to recurrence of the meningitis, but we now realize that these phenomena are caused by the intrathecal administration of penicillin. The syndrome has been named "meningeal pseudoexacerbation." Our experiments were designed to determine: *1.* the factors which might be responsible for eliciting the syndrome during intrathecal penicillin therapy, and *2.* to find a method for differentiating between true and pseudoexacerbations.

ETIOLOGICAL FACTORS. AND SYMPTOMS.

Any of the following factors must be considered as a cause of the symptoms: *1.* spinal puncture and withdrawal of CSF, *2.* insufflation of air during spinal puncture, *3.* secondary infection, *4.* the sodium chloride in which the penicillin is in solution, and *5.* penicillin.

The untoward effects of lumbar puncture are commonly known: injury to cauda, local pain, paresthesia and neuralgia in the legs may occur and last for several days. If CSF is withdrawn, the disturbances of hydrostatic equilibrium may be associated with prostration, nausea, vomiting, headache, backpain (Birkholz[1]), and leucocytosis (Porta and Foat, and

*From St. John Hospital of Budapest City.

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Roizin[2,3]). The complaints are accompanied by no sytochemical changes. It is well known that the insufflation of air into the spinal canal may be followed by headache, nausea, vomiting and fever.

The needle employed in the puncture is passed through the skin, which is difficult to sterilize, and if serial punctures are performed the likelihood of a secondary infection is increased. Secondary infections attending lumbar puncture have been reported by Mallaret and Reilly,[4] and Smith, Duthie and Cairns.[5] The latter authors found bacteria different from those previously present in the CSF in 11 cases. The seconary infections were unaccompanied by a cellular reaction, and their course was benign. Infections with B. pyocyaneus are particularly dangerous (Botterel and Wagner[6]). Penicillin in a closed vial may be contaminated and is more apt to be so if the vial has been opened.

The possibility of the physiological sodium chloride causing untoward symptoms has not been described, and it was my desire to clarify this problem. Patients suffering from schizophrenia or epilepsy, who had a normal CSF and no fever for a week, were chosen for study. Absolute surgical asepsis was observed. The injected fluid had the temperature of 37° C. Various quantities, 1 cc., 5 cc. and 10cc., were injected into the spinal canal and cisterna, care being taken to keep air out of the syringe. We looked for any difference in the reaction between the intralumbar and intracisternal routes of the administration. The CSF originating from the cisterna was examined separately from that obtained from the spinal canal. Ten patients were given normal saline in the spinal canal and eight received it intracisternally.

Fever was one symptom encountered. The peak of 38° C. occurred five to eight hours following injection. It gradually receded but low grade pyrexia was still evident on the second day. Nine patients complained of headache and three of nausea and vomiting. Increased pressure of the CSF and pleocytosis were invariably present, and in seven cases protein content was increased. Ninety per cent of the cells were leucocytes. An increase in quantity of the saline injected magni-

fied the CSF changes. When equal quantities were injected intracisternally and in the lumbar region greater changes were noted in those cases in which intracisternal administration was carried out.

Rammelkanp[7] and Keifer, Johnson and Walker,[8] Rosenberg and Arling,[9] Cairns, Lewin, Deuthie and Smith[10] contend that the reactive symptoms following intrathecal penicillin administration are due to high concentration of the drug, while Cairns[11] and Forray[12] attribute the symptoms to chemical contamination. We endeavor to demonstrate the conditions under which these symptoms occurred in normal individuals and the time of occurrence in patients with meningitis.

Twenty-three individuals with normal CSF received various quantities of penicillin intralumbarly[13] and intracisternally.[10]

The temperature rose to 38° C., lasted for some hours and did not become normal before the second day. Three patients complained of vomiting and 15 of headache. The pressure and cell count of the CSF was increased. The protein content was elevated in 11 cases and 90 per cent of the cells were leucocytes. The sugar concentration was determined in four cases and found to be diminished in two; in comparison with cell count the protein content was relatively low.

In four patients examination of the CSF was again carried out 72 hours after the administration of penicllin. Cell counts were six, 17, 23 and 124. Twenty to 30 per cent were leucocytes. Protein concentration was normal, and in a few cases a subnormal temperature persisted for 48 to 72 hours. These data have not been taken into consideration in the table. The penicillin was given in 5,000 to 30,000 unit doses and the reactive symptoms were independent of quantity administered. Table I shows the correlation between the concentration of the solution and the symptoms.

The reactive symptoms are milder if the concentration of penicillin is higher.

TABLE I.

CORRELATION OF REACTION AND CONCENTRATION OF PENICILLIN.

Concentration: Penicillin in 1 cc. of NaCl	Number and Percentage of Cases	Cell Count in the C.S.F. Under 400	Cell Count in the C.S.F. Over 400	Temperature /°C./ Normal	Temperature /°C./ 37-38	Temperature /°C./ Over 38
1,000 units	13	4	9	4	5	4
	100%	30.7	69.3	30.7	38.6	30.7
10,000 units	10	6+	3	4	2	4
	100%	60	30	40	20	40

+ = not to be evaluated: 1/10%/.

In three instances 20,000 units of penicillin dissolved in 2 cc. of CSF removed from cisterna were injected. Two of these patients were asymptomatic, whereas the third complained of fever, headache and vomiting and exhibited serious CSF changes. We assumed that this preparation was chemically contaminated.

In Table II the symptoms following the intralumbar and intracisternal injection of sodium chloride and penicillin solution are summarized.

The symptoms of pyrexia, headache, nausea, vomiting, dizziness and prostration were more conspicuous following intracisternal injection. Pleocytosis was also more marked than after intralumbar administration.

Comparison of the Lumbar and Cisternal CSF Taken Simultaneously.

This examination was performed with 17 patients, eight of whom received intralumbar, and nine intracisternal penicillin. In all cases save one the cell count and protein count of the spinal fluid were higher than those of the cisternal fluid. On the average the spinal cell count was 2.5 times and the protein content was 5.5 times the cisternal value. Three patients had a normal protein content in the cisternal fluid while that of the spinal fluid was increased.

In correlating cell count and protein content it was noted that in 11 out of 31 cases (35.4 per cent) the spinal fluid

TABLE II. DEPENDENCE OF REACTION ON THE ROUTE OF ADMINISTRATION.

Route	Number and Percentage of Cases	General symptoms:							C.S.F.					
		No Complaint	Headache	Vomiting	Temperature /°C./ Normal	37-38	Over 38	+	Cell Count Under 400	Over 400	++	Protein Under 60 mg. %	Over 60 mg. %	+++
Lumbar	23	13	8	2	8	7	5	3	12	10	1	14	6	3
	100%	56.7	34.7	8.7	34.7	30.4	21.7	13.2	52.1	43.4	4.5	60.8	26	13.2
Cisternal	18	3	11	4	2	3	13		7	11		12	6	
	100%	16.6	61.2	22.2	11.1	16.6	72.2		38.8	61.2		66.6	33.4	

+ = not to be evaluated.

exhibited a normal protein content and high cell count. The same correlation was found in the cisternal CSF in 13 out of 25 instances (58.3 per cent).

REACTIVE SYMPTOMS IN MENINGITIS.

During the period 1946-1948, 31 meningitis patients in our hospital were treated with penicillin. In the most severe stage of the disease intrathecal penicillin therapy was not attended by any reaction; reactive symptoms did not appear until the recovery process had started. Pseudoexacerbations were observed in eight cases; in six of them the symptoms occurred when the spinal CSF cell count ranged from 30 to 160 and the protein count from 35 to 65 mg. per cent. In the seventh patient the cell count was 320 and the protein content 75. In the eighth case the reaction began when the spinal fluid cell count was 1,600 and the protein content 45 to 60, while the cisternal fluid cell count was 200 and the protein content 30. Reactive symptoms coincide with cell counts of 100 to 200 and a protein content of less than 45 to 60 mg. when the recovery from meningitis has started.

Examinations to Differentiate Between True and Pseudoexacerbations.

In 1946, our experience with penicillin therapy in meningitis was limited and we were unaware of the reactive symptoms due to its use. For this reason, in our first cases we were puzzled when the meningeal pseudoexacerbations occurred. At first we thought that the meningitis had become worse and continued to administer penicillin for a time while the symptoms became more marked.

Case 1: A boy, age 13, was admitted Sept. 29, 1946. Six days prior to admission he developed pain in right ear and spontaneous discharge on the following day. The temperature was 39° C. Everything he ate was vomited, and he lay motionless. He had lost consciousness on the morning of admission and had fully developed meningeal symptoms and purulent spinal fluid; 20,000 units of penicillin were given immediately. The left ear was intact. On the right side there was profuse discharge and the drum was swollen and hyperemic. Radical mastoidectomy was performed with wide exposure of the dura of the middle and posterior fossa. There was extensive pneumatization reaching to the zygomatic root and posterior to the lateral sinus. The cells were full of pus, and the dura of the middle fossa was markedly hyperemic. Transfusion, cardiac stimulants and penicillin were administered.

Return to consciousness and general improvement were noted on the first postoperative day. That evening high fever, delirium, headache, nausea, nuchal rigidity and positive Kernig's sign appeared and the CSF pointed to deterioration. Recovery was rapid when intrathecal penicillin injection was halted.

Case 2: A 25-year-old woman was admitted on Nov. 14, 1946. She had been treated for pulmonary tuberculosis with pneumothorax for two years. For four months prior to admission she had suffered from purulent tuberculous otitis media. For three days the fever had ranged from 38 to 40° C. and auricular and retroauricular pain, chills, fever, nausea, vomiting and headache were present.

State on admission: There was neck rigidity. Kernig's and Brudzinski's signs were absent. The skin was hyperesthetic, the tongue and pharynx dry and furred, the reflexes spastic. Ophthalmoscopy revealed dilated veins, sharply limited papillae and normal pupillary reaction on both sides (Dr. Mócsy). Spinal fluid was clear and under increased pressure. The right eardrum was intact. On the left side a purulent discharge was found in the external meatus, the drum was swollen and hyperemic. Landmarks were absent. There was an anterior inferior perforation with pulsating discharge. The mastoid and jugular vein areas were tender to pressure. On Nov. 15 antrotomy with exposure of the lateral sinus was done. The cells were medium sized and near the apex they were lined by intact mucosa, whereas at the antral region the mucosa was swollen and the cells were filled with serohemorrhagic and seropurulent secretion from which streptococci were cultivated. The anterior sinus wall was grayish and thickened, and the lumen contained a red thrombus which was removed by incision and suction. Penicillin was given, first intramuscularly three times daily (20,000 units per dose). After CSF examination intrathecal administration was begun. On the first postoperative day the neck was mobile, and the general condition much improved; but later nausea, vomiting, prostration and rigidity of the neck appeared. The spinal fluid was repeatedly hemorrhagic, so occipital puncture was performed, and penicillin was injected into the cisterna (20,000 units in 20 cc. of sodium chloride). Prostration, vomiting and cervical rigidity became more marked. Quick recovery followed cessation of penicillin therapy.

In this case the intrathecal administration was thoroughly superfluous, the CSF being normal; however, the laboratory reports had not been received before the second day. Irritative symptoms suggested the presence of an intracranial complication, hence we were reluctant to discontinue intrathecal administration. Finally, recovery immediately followed its omission.

It was these cases that induced us to perform experiments with intrathecal penicillin administration in so-called normal cases. As may be seen, the reactive symptoms ceased in 48 to 72 hours. For this reason the problem can best be solved by observing the pseudoexacerbation after the omission of penicillin. This procedure would be dangerous in some patients, so efforts were made to find a method by which true recurrences could safely be differentiated from pseudoexacerbations.

It was stated that, after the intrathecal administration of penicillin, the cell count of the spinal fluid was 2.5 times its protein content, 1.5 times as high as the values found in the cisternal fluid. We further found that the protein content of the spinal fluid was pathologically increased. In some instances this was proven by normal protein content of the cisternal CSF. In an effort to apply these phenomena clinically, we performal simultaneous tappings in dubious cases. Results of simultaneous punctures cannot be evaluated in meningitis unless the findings are compared with the results of previous punctures.

The following cases exemplify the differentiation between recurrence and pseudoexacerbation by comparing the findings of the spinal and cisternal CSF taken simultaneously.

Case 3: A man of 46 was admitted June 12, 1947. Symptoms of ear pain; fever, retroauricular pain and no discharge were experienced three weeks before entry. Later the pain ceased but the hearing remained impaired. Five days prior to admission, fever, ear pain and retroauricular pain recurred. Headache, chills, fever and vomiting followed two days later. He was comatose on admission. Sulfathiazole was given. On the following day the patient regained consciousness while meningeal symptoms persisted. The right tympanic membrane was swollen and hyperemic; landmarks were obliterated. The mastoid region was tender. Intramuscular and intrathecal penicillin were introduced. Antrotomy was done on June 14. Pneumatization was microcellular, and the mucosa was hyperemic and edematous. The periantral and perizygomatic cells contained much pus.

Early omission of intrathecal therapy resulted in a true recrudescence as seen from the findings of the CSF taken on June 20. The cell content of the cisternal fraction was higher than that of the spinal CSF and the protein concentration of both fractions was increased. Sugar content was diminished. Ninety-eight per cent of the cells were polymorphonuclear leucocytes. Bacteria were demonstrated in the CSF. On June 23, CSF findings showed undoubted improvement of the meningeal process: the cell count of the spinal fraction exceeds many times that of the cisternal CSF while the protein content of the cisternal fluid is nearly normal. The sugar level was higher in the cisternal than in the spinal fraction, which is the normal state. The percentage of lymphocytes gradually increased. We then injected a last dose of penicillin (10,000 units) into the cisterna. The following day penicillin was stopped in spite of the increasing cell count. The patient recovered.

The problem was still more difficult in the next case. Though the simultaneous punctures pointed to a pseudoexacerbation, we continued the intrathecal therapy because we did not hold the symptoms reliable.

Case 4: A man, aged 24, was admitted Dec. 5, 1947, with the history of recurrently discharging ears since the age of three years. The hearing had been failing progressively. He had two attacks of meningitis, one in 1926 and one in 1944. Two weeks before admission he developed an acute

cold. Both ears became full and tenderness appeared in the retroauricular region. Relief followed in three days and he was apparently well. On the day of admission, chills, fever, headache and vomiting occurred.

State on admission: Meningeal symptoms, clear sensorium. Thick fluid in the right external ear canal. Tympanic membrane swollen and red, with obliteration of anatomical detail. No mastoid tenderness was present. Intramuscular and intrathecal penicillin were given.

On Dec. 7, intralumbar and intrathecal penicillin injections were given. The patient then complained of malaise and headache, and vomiting ensued. The cell count of the cisternal fraction was 200 as compared with 1,600 in the spinal fraction and the protein concentration was nearly normal. The high 9,600 cell count obtained the following day was undoubtedly due to the intrathecal administration of penicillin. Being ignorant of the diagnostic value of the comparative examination of the spinal and cisternal CSF, we continued the intrathecal penicillin until a cell count of 30 was obtained.

Case 5: A 36-year-old man was admitted Dec. 6, 1947. His complaints of thoracic pain, cough, fever and headache have been evident for one week.

State on admission: Periodic delirium, meningeal symptoms, bilateral pneumonia, jaundice. No otolaryngologic abnormality was noted. Intramuscular and intrathecal penicillin therapy was introduced.

Following penicillin therapy the fever receded, and the CSF exhibited improvement. On the third day the fever returned, and nausea and headache were experienced. On Dec. 10, simultaneous punctures were done. The CSF showed the signs of uneventful healing, but the general symptoms became worse. Believing the symptoms to be due to intrathecal penicillin, this route was abandoned while intramuscular doses were raised. Sulfadiazine was started. CSF findings on Dec. 12 corresponded to the reactive symptoms elicited by intrathecal penicillin therapy. This assumption was confirmed by the fact that the patient felt well and was afebrile on Dec. 13. On Dec. 14, he again developed hyperpyrexia. Recurrence was assumed though meningeal symptoms were absent. For this reason simultaneous punctures were performed on Dec. 15, and penicillin was injected into the cisterna; however, the findings indicated recovery. Thus the findings of Dec. 12 could have been due to the reaction to intrathecal penicillin.

An experiment was then performed on a 72-year-old woman who was improving following a pneumococcic meningitis. On the sixth day following the omission of intrathecal penicillin therapy we injected 10 cc. of sterile physiologic NaCl into the spinal canal. In the CSF taken before the injection the cell count was 11 and the protein content 90 mg. per cent. Twenty-four hours later the cell count was 126 in the spinal fluid and 39 in the cisternal fraction. Protein content of both samples was 75 mg. per cent.

Meningeal pseudoexacerbation was encountered in three cases. In two the presence of pseudoexacerbation was obvious,

whereupon the intrathecal therapy was discontinued. In the third patient there was doubt and the diagnosis was made by means of simultaneous tappings.

CONCLUSIONS.

On the basis of the data of literature and our own experimental findings it could be stated that the reaction due to intrathecal administration of penicillin may be due to the separate or the simultaneous action of several factors. Spinal puncture and withdrawal of CSF led to general symptoms only, whereas the injection of air, sodium chloride or penicillin may give rise to changes in the CSF also. The symptoms following the intralumbar and intracisternal administration, respectively, are not quite identical. In the majority of cases intracisternal injections are followed by high fever and marked general complaints and a higher degree of pleocytosis. From the viewpoint of the evaluation of the symptoms of reaction and in order to reduce reaction to intrathecal penicillin, the following principles should be taken into consideration: aseptic technique must be observed; the vial containing penicillin should not be opened prior to the intrathecal injection; penicillin is to be dissolved in the CSF. If physiologic sodium chloride is to be applied as the solvent, the right proportion is 10,000 units per cc. When the process is subsiding the intralumbar route should be chosen without insufflation of air except in cases where adhesions are to be prevented.

In the course of a meningitis reactive symptoms to intrathecal penicillin administration do not occur. This is understandable, considering the fact that while the meningitis persists, two stimuli act at the same time: the infection and the penicillin. When the irritation due to the infection persists (dilation of vessels, emigration of cells) the administration of penicillin has no sequelae; however, when healing begins, the introduction of a new irritant is followed by reaction. Intensity of reactive symptoms also depends upon the age of the patient and the duration of the meningeal process. In the presence of old age or long standing disease the functional capacity of the tissues is restored with difficulty; therefore,

the protein content of the CSF, due perhaps to the lasting damage of the vessel wall, may be high for several months after the clinical recovery.

When a penicillin reaction appears during recovery it has a prognostic value. It points to the onset of recovery. It may be inferred from this fact that the intrathecal administration of penicillin should be omitted when these reactive symptoms ensue; that is, when the cell count of the CSF does not exceed 100 to 200.

Pseudoexacerbation can best be distinguished from a true recrudescence by the temporary interruption of the intrathecal penicillin therapy. Reactive symptoms recede in 24 to 72 hours. If the interruption of therapy is considered hazardous the two conditions can be differentiated by the examination of the spinal and cisternal fractions of the CSF taken simultaneously. True recurrence may be assumed if the cell count of the cisternal fluid is many times that of the spinal sample, the protein concentration increased in both fractions and a predominance of polymorphonuclear leucocytes (over 90 per cent). The pathogenic microorganisms do as a rule reappear; however, their absence is not conclusive evidence against a true recrudescence. Pseudoexacerbation on the other hand is characterized by a higher cell count in the spinal fraction than in the cisternal sample and a closely normal protein content in both; if, however, there is an increase in proteins it occurs in the lumbar fraction. The sugar level is normal or slightly reduced, and its value is somewhat higher in the cisternal sample. Though polymorphonuclear leucocytes may be increased, there are fewer than 90 per cent. If bacteria are present they are saprophytes.

SUMMARY.

1. The intrathecal administration of sodium chloride is followed by symptoms of meningeal irritation. Similar effect is exerted by penicillin.

2. The symptoms are independent of the quantity of penicillin, but they are milder if the drug is injected in a more concentrated solution.

3. Penicillin should be dissolved in the CSF or if sodium chloride is employed as the dissolvent, the right proportion is 10,000 units per cc.

4. The intrathecal (lumbar or cisternal) administration of penicillin is followed by a change in the cell count and protein content. The cell count and protein content of the spinal fluid is 2.5 times and 1.5 times, respectively, higher than the corresponding values of the cisternal fluid. The dissociation of the cell count and protein content is less marked in the spinal fluid than in the cisternal one.

5. Intralumbar injections are usually followed by less intensive symptoms than are intracisternal injections.

6. Intrathecal administration of penicillin has no sequelae in meningitis. A reaction during recovery indicates that the process of healing has started. At this time (cell count 100 to 200) intrathecal penicillin therapy is useless or harmful.

7. Recurrences and pseudoexacerbations may be differentiated by temporarily interrupting intrathecal penicillin therapy or by simultaneously examining cisternal and lumbar fractions of CSF. The cell count, protein level and sugar content serve as guides in the differential diagnosis.

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The Directory issue of *Graphics*, the official publication of the Association of Medical Illustrators, contains the name, address, training, professional experience and reference to major published work of each member. Other information pertaining to the profession is included.

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As of April 1, 1950.

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Gem Hearing Aid Model V-35.
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Maico Type K; Maico Atomeer; Maico UE-Atomeer.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

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Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Silvertone Model 103BM.

Distributor: Sears-Roebuck & Co., Chicago, Ill.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925.

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Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
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Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.
Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

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Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.
Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.
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Time: 6:00 P.M., fourth Monday of each month from September to May, inclusive.

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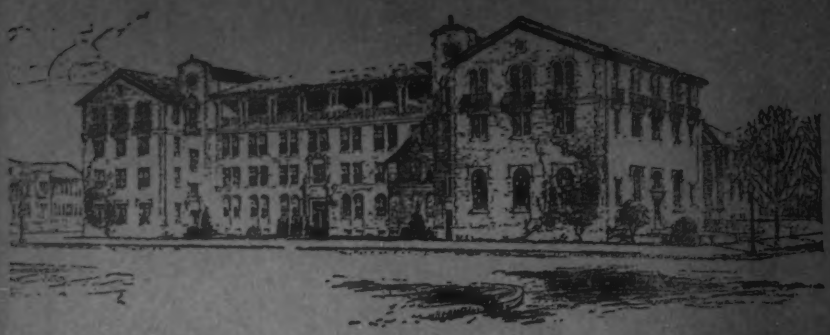

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